Correspondence

Detection of anti-dsDNA as a diagnostic tool

SIR, We read with interest the paper by Drs Swaak and Smeenk on the detection of antibodies to double-stranded deoxyribonucleic acid (anti-dsDNA) as a diagnostic tool. Of 441 patients without systemic lupus erythematosus (SLE) but with anti-dsDNA no fewer than 304 (69%) developed SLE within one year as judged by the preliminary classification criteria of the American Rheumatism Association (ARA). Longer follow up showed a cumulative incidence of SLE of 85%. We have found different results in a group of patients who, in contradistinction to the Dutch study cases, did not have antinuclear antibodies (ANA) on conventional indirect immunofluorescent testing, despite high titres of anti-dsDNA.

By reference to the anti-dsDNA results file in the pathology service laboratory covering the period 1976-83, we have identified 20 patients (12 female, eight male; mean age 51 years) who were followed up for at least a year and who had anti-dsDNA titres in excess of 30 U/ml (anti-DNA kit, Amershams International) on two or more occasions. Tests for ANA with serum diluted 1:16 on a substrate of rat liver slices were negative on every occasion for every patient included in the study. Two patients subsequently developed ANA at 20 and 25 months respectively after the anti-dsDNA were first noted. The other 18 patients remained consistently ANA negative and were tested on three to 12 occasions (mean 5.1).

Systemic lupus erythematosus

SIR, The report of Soppi, Eskola, and Lehtonen on identical twins discordant over 20 years for clinically

evident SLE adds to our understanding of the importance of both genetic and environmental factor(s) in the expression of this disease. To date it remains unknown whether the genetic factor(s) is requisite and the environmental factor only stimulatory or whether a certain combined quantity (more or less of one and/or the other) of the two sufficient to reach an expressive threshold is critical.

In the twins described it is of interest that the clinically unaffected sibling had a persistently raised erythrocyte sedimentation rate (ESR), for although the presence of antinuclear antibodies and other protein aberrations, including low complement levels, need not be associated with active disease, a raised ESR, though non-specific, usually suggests the presence of active inflammation or tissue damage. Could something subtle have been missed? Twenty years' observation argues against this notion, but it is not necessarily reassuring for the future. I look forward to a follow-up report 10 years from now.

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Reference

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